```
=> s G-protein coupled receptors and fat metabolism
L1            0 G-PROTEIN COUPLED RECEPTORS AND FAT METABOLISM

=> s G-protein coupled receptor? and fat metabolism
L2            7 G-PROTEIN COUPLED RECEPTOR? AND FAT METABOLISM

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3            3 DUP REM L2 (4 DUPLICATES REMOVED)

=> d l3 1-3 ibib ab

L3   ANSWER 1 OF 3     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:   2003259287     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 12784853
TITLE:              Physiologic melatonin concentration, omega-3 fatty acids,
                    and conjugated linoleic acid inhibit fatty acid transport
                    in rodent hind limb skeletal muscle in vivo.
AUTHOR:             Dauchy Robert T; Blask David E; Sauer Leonard A; Davidson
                    Leslie K; Krause Jean A; Smith Laura C; Dauchy Erin M
CORPORATE SOURCE:   Laboratory of Experimental Neuroendocrinolgy/Oncology,
                    Bassett Research Institute, Cooperstown, New York
                    13326-1394, USA.
CONTRACT NUMBER:    R01CA76197 (NCI)
SOURCE:             Comparative medicine, (2003 Apr) 53 (2) 186-90.
                    Journal code: 100900466. ISSN: 1532-0820.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200312
ENTRY DATE:         Entered STN: 20030606
                    Last Updated on STN: 20031217
                    Entered Medline: 20031216
AB   Melatonin (MLT), the circadian neurohormone secreted by the pineal gland
     in mammals during darkness, eicosapentanoic acid (EPA), and conjugated
     linoleic acid (CLA) have established regulatory roles in cancer growth.
     Investigations in our laboratory have indicated that these agents inhibit
     fatty acid (FA) transport by tumors and several sub-types of white adipose
     tissue via inhibitory G protein-coupled
     receptor mechanisms.  Skeletal muscle constitutes over 45% of
     human body mass and plays an important role in cancer cachexia and
     obesity-related diseases.  Since fatty acid oxidation is a major source of
     energy for this tissue, we tested the hypothesis that physiologic MLT
     levels, EPA, or CLA injected intravenously, inhibit FA uptake in rat
     skeletal muscle in vivo.  We used a surgical technique for catheterizing
     the femoral vein in rats that allows rapid blood collection from the
     entire hind limb, while ensuring continuous blood flow to the tissue.
     Blood acid/gas tensions and hematocrit were monitored and remained
     constant during the course of each experiment.  The MLT, EPA, and CLA
     inhibited FA uptake by the tissue and lowered cAMP values.  Glucose uptake
     and glycerol production in the hind limb were not affected.These
     investigations suggest a novel role for MLT, omega-3 FAs, and CLA in the
     regulation of FA transport and fat metabolism in
     skeletal muscle.

L3   ANSWER 2 OF 3     MEDLINE on STN                    DUPLICATE 2
ACCESSION NUMBER:   2000127771     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 10666005
TITLE:              GIP biology and fat metabolism.
AUTHOR:             Yip R G; Wolfe M M
CORPORATE SOURCE:   Department of Medicine, Boston University School of
                    Medicine, Boston Medical Center, MA 02118, USA.
SOURCE:             Life sciences, (2000) 66 (2) 91-103.  Ref: 95
                    Journal code: 0375521. ISSN: 0024-3205.
```

PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000309
Last Updated on STN: 20000309
Entered Medline: 20000222

AB The gastrointestinal hormone, gastric inhibitory polypeptide (GIP), is synthesized and released from the duodenum and proximal jejunum postprandially. Its release depends upon several factors including meal content and pre-existing health status (ie. obesity, diabetes, age, etc.). It was initially discovered and named for its gastric acid inhibitory properties. However, its more physiologically relevant role appears to be as an insulinotropic agent with a stimulatory effect on insulin release and synthesis. Accordingly, it was later renamed glucose-dependent insulinotropic polypeptide because its action on insulin release depends upon an increase in circulating levels of glucose. GIP is considered to be one of the principle incretin factors of the enteroinsular axis. The GIP receptor is a **G-protein-coupled receptor** belonging to the family of secretin/VIP receptors. GIP receptor mRNA is widely distributed in peripheral organs, including the pancreas, gut, adipose tissue, heart, adrenal cortex, and brain, suggesting it may have other functions in addition to the ones mentioned above. An overactive enteroinsular axis has been suggested to play a role in the pathogenesis of diabetes and obesity. In addition to stimulating insulin release, GIP has been shown to amplify the effect of insulin on target tissues. In adipose tissue, GIP has been reported to (1) stimulate fatty acid synthesis, (2) enhance insulin-stimulated incorporation of fatty acids into triglycerides, (3) increase insulin receptor affinity, and (4) increase sensitivity of insulin-stimulated glucose transport. In addition, although controversial, lipolytic properties of GIP have been proposed. The mechanism of action of GIP-induced effects on adipocytes is unknown, and it is unclear whether these effects of GIP on adipocytes are direct or indirect. However, there is now evidence that GIP receptors are expressed on adipocytes and that these receptors respond to GIP stimulation. Given the location of its release and the timing of its release, GIP is an ideal anabolic agent and expanding our understanding of its physiology will be needed to determine its exact role in the etiology of diabetes mellitus and obesity.

L3 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:818119 HCAPLUS
DOCUMENT NUMBER: 132:117676
TITLE: GIP biology and **fat metabolism**
AUTHOR(S): Yip, Rupert G. C.; Wolfe, M. Michael
CORPORATE SOURCE: Section of Gastroenterology, Department of Medicine, Boston Medical Center, Boston University School of Medicine, Boston, MA, 02118, USA
SOURCE: Life Sciences (1999), Volume Date 2000, 66(2), 91-103
CODEN: LIFSAK; ISSN: 0024-3205
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review, with 94 refs. The gastrointestinal hormone, gastric inhibitory polypeptide (GIP), is synthesized and released from the duodenum and proximal jejunum postprandially. Its release depends upon several factors including meal content and pre-existing health status (ie. obesity, diabetes, age, etc.). It was initially discovered and named for its gastric acid inhibitory properties. However, its more physiol. relevant role appears to be as an insulinotropic agent with a stimulatory effect on insulin release and synthesis. Accordingly, it was later renamed glucose-dependent insulinotropic polypeptide because its action on insulin

release depends upon an increase in circulating levels of glucose. GIP is considered to be one of the principle incretin factors of the enteroinsular axis. The GIP receptor is a **G-protein-coupled receptor** belonging to the family of secretin/VIP receptors. GIP receptor mRNA is widely distributed in peripheral organs, including the pancreas, gut, adipose tissue, heart, adrenal cortex, and brain, suggesting it may have other functions in addn. to the ones mentioned above. An overactive enteroinsular axis has been suggested to play a role in the pathogenesis of diabetes and obesity. In addn. to stimulating insulin release, GIP has been shown to amplify the effect of insulin on target tissues. In adipose tissue, GIP has been reported to (1) stimulate fatty acid synthesis, (2) enhance insulin-stimulated incorporation of fatty acids into triglycerides, (3) increase insulin receptor affinity, and (4) increase sensitivity of insulin-stimulated glucose transport. In addn., although controversial, lipolytic properties of GIP have been proposed. The mechanism of action of GIP-induced effects on adipocytes is unknown, and it is unclear whether these effects of GIP on adipocytes are direct or indirect. However, there is now evidence that GIP receptors are expressed on adipocytes and that these receptors respond to GIP stimulation. Given the location of its release and the timing of its release, GIP is an ideal anabolic agent and expanding our understanding of its physiol. will be needed to det. its exact role in the etiol. of diabetes mellitus and obesity.

REFERENCE COUNT: 95 THERE ARE 95 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	13.85	14.06

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-0.75	-0.75

STN INTERNATIONAL LOGOFF AT 13:52:58 ON 22 FEB 2006

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L4: Entry 2 of 2 File: DWPI May 26, 2005

DERWENT-ACC-NO: 2005-403353
DERWENT-WEEK: 200541

TITLE: Identification of a compound for treating a disorder in fat metabolism, comprises contacting a compound with a cell, and determining a G-protein stimulatory subunit expression level or activity in the cell

INVENTOR: LEE, Y

PATENT-ASSIGNEE:

ASSIGNEE	CODE
ACAD SINICA	SININ

PRIORITY-DATA: 2002US-0211423 (August 2, 2002), 2004US-0981237 (November 4, 2004)

Search Selected Search ALL Clear

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 20050112668 A1	May 26, 2005		006	C12Q001/68

APPLICATION-DATA:

PUB-NO	APPL-DATE	APPL-NO	DESCRIPTOR
US20050112668A1	August 2, 2002	2002US-0211423	Div ex
US20050112668A1	November 4, 2004	2004US-0981237	

INT-CL (IPC): A61 K 48/00; C12 Q 1/68

RELATED-ACC-NO: 2004-156251

ABSTRACTED-PUB-NO: US20050112668A
BASIC-ABSTRACT:

NOVELTY - Identification of a compound for treating a disorder in fat metabolism, comprises contacting a compound with a cell, and determining a G-protein stimulatory subunit (Gsa) expression level or activity in the cell, where the Gsa expression level or activity in the presence of the compound, if different from that in the absence of the compound, indicates that the compound is a candidate for treating a disorder in fat metabolism.

USE - The invention deals with the identification of a compound for treating a disorder in fat metabolism.

CHOSEN-DRAWING: Dwg.0/0

TITLE-TERMS: IDENTIFY COMPOUND TREAT DISORDER FAT METABOLISM COMPRISE CONTACT COMPOUND CELL DETERMINE PROTEIN EXPRESS LEVEL ACTIVE CELL

DERWENT-CLASS: B04 D16

CPI-CODES: B04-F02; B04-K01Y; B11-C08E1; B11-C08E7; B11-C10; B12-K04E1; B14-E11; B14-E11A; B14-F06; B14-L01; B14-L06; D05-H08; D05-H09;

CHEMICAL-CODES:

Chemical Indexing M6 *01*
Fragmentation Code
M905 P617 P714 P731 P814 P831 Q233 Q505 R515 R521
R614 R627 R633 R637 R639

SECONDARY-ACC-NO:
CPI Secondary Accession Numbers: C2005-124629

Previous Doc Next Doc Go to Doc#

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=> file medline hcaplus biosis embase uspatfull
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                        ENTRY    SESSION
FULL ESTIMATED COST                                      0.21       0.21

FILE 'MEDLINE' ENTERED AT 13:35:53 ON 22 FEB 2006

FILE 'HCAPLUS' ENTERED AT 13:35:53 ON 22 FEB 2006
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FILE 'USPATFULL' ENTERED AT 13:35:53 ON 22 FEB 2006
CA INDEXING COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

=> s G-protein coupled receptor protein and fat metabolism
L1            1 G-PROTEIN COUPLED RECEPTOR PROTEIN AND FAT METABOLISM

=> s G-protein coupled receptors and fat metabolism
L2           37 G-PROTEIN COUPLED RECEPTORS AND FAT METABOLISM

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3           37 DUP REM L2 (0 DUPLICATES REMOVED)

=> s l3 and modulator?
L4           20 L3 AND MODULATOR?

=> s l4 and mRNA
L5           16 L4 AND MRNA

=> s l5 and microarray
L6            4 L5 AND MICROARRAY

=> d l6 1-4 ibib ab

L6   ANSWER 1 OF 4  USPATFULL on STN
ACCESSION NUMBER:       2006:17559  USPATFULL
TITLE:                  Proteins involved in the regulation of energy
                        homeostasis
INVENTOR(S):            Eulenberg, Karsten, Bovenden, GERMANY, FEDERAL REPUBLIC
                        OF
                        Meise, Martin, Gottingen, GERMANY, FEDERAL REPUBLIC OF
                        Molitor, Andreas, Gottingen, GERMANY, FEDERAL REPUBLIC
                        OF
                        Steuernagel, Arnd, Gottingen, GERMANY, FEDERAL REPUBLIC
                        OF

                             NUMBER        KIND    DATE
                        --------------- ------ --------
PATENT INFORMATION:     US 2006015951      A1   20060119
APPLICATION INFO.:      US 2003-531036     A1   20031014  (10)
                        WO 2003-EP11352         20031014
                                                20050412  PCT 371 date

                               NUMBER         DATE
                        ------------------ --------
PRIORITY INFORMATION:   EP 2003-2022880    20021014
                        EP 2003-2023560    20021022
```

EP 2003-2024747 20021106
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ROTHWELL, FIGG, ERNST & MANBECK, P.C., 1425 K STREET, N.W., SUITE 800, WASHINGTON, DC, 20005, US
NUMBER OF CLAIMS: 35
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 61 Drawing Page(s)
LINE COUNT: 3439
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention discloses novel uses for energy homeostasis regulating proteins and polynucleotides encoding these in the diagnosis, study, prevention, and treatment of metabolic diseases and disorders.

L6 ANSWER 2 OF 4 USPATFULL on STN
ACCESSION NUMBER: 2005:130659 USPATFULL
TITLE: Compositions and methods for treating inflammatory disorders
INVENTOR(S): Cimbora, Daniel, Salt Lake City, UT, UNITED STATES
Heichman, Karen, Salt Lake City, UT, UNITED STATES
Bartel, Paul, Salt Lake City, UT, UNITED STATES
Mauck, Kimberly, Sandy, UT, UNITED STATES
Bush, Angie, Sandy, UT, UNITED STATES
PATENT ASSIGNEE(S): Myriad Genetics, Incorporated, Salt Lake City, UT, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005112118	A1	20050526
APPLICATION INFO.:	US 2003-690276	A1	20031020 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-727384, filed on 1 Dec 2000, ABANDONED Continuation-in-part of Ser. No. US 2002-35344, filed on 4 Jan 2002, PENDING Continuation-in-part of Ser. No. US 2002-35343, filed on 4 Jan 2002, ABANDONED Continuation-in-part of Ser. No. US 2002-99924, filed on 14 Mar 2002, PENDING Continuation-in-part of Ser. No. US 2002-100503, filed on 18 Mar 2002, PENDING Continuation-in-part of Ser. No. US 2001-14814, filed on 14 Dec 2001, ABANDONED Continuation-in-part of Ser. No. US 2001-24599, filed on 21 Dec 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-168377P	19991202 (60)
	US 1999-168379P	19991202 (60)
	US 2000-185056P	20000225 (60)
	US 2001-259571P	20010104 (60)
	US 2001-259572P	20010104 (60)
	US 2001-276179P	20010315 (60)
	US 2001-307233P	20010723 (60)
	US 2001-277013P	20010319 (60)
	US 2000-255063P	20001214 (60)
	US 2000-256986P	20001221 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MYRIAD GENETICS INC., INTELLECUTAL PROPERTY DEPARTMENT, 320 WAKARA WAY, SALT LAKE CITY, UT, 84108, US
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 34 Drawing Page(s)
LINE COUNT: 13483
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Protein complexes are provided comprising at least one interacting pair of proteins. The protein complexes are useful in screening assays for

identifying compounds effective in modulating the protein complexes, and in treating and/or preventing diseases and disorders associated with the protein complexes and/or their constituent interacting members.

L6 ANSWER 3 OF 4 USPATFULL on STN
ACCESSION NUMBER: 2004:204986 USPATFULL
TITLE: Polynucleotide and polypeptide **fat metabolism** regulators and uses thereof
INVENTOR(S): Ruvkun, Gary, Newton, MA, UNITED STATES
Ashrafi, Kaveh, San Francisco, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004158879	A1	20040812
APPLICATION INFO.:	US 2003-617351	A1	20030710 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-395159P	20020711 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: CLARK & ELBING LLP, 101 FEDERAL STREET, BOSTON, MA, 02110
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 26 Drawing Page(s)
LINE COUNT: 8116
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB In general, this invention relates to nucleic acid and amino acid sequences involved in **fat metabolism** regulation and the use of these sequences as targets for the diagnosis, treatment, and prevention of obesity and obesity-related diseases. In addition, the invention relates to screening methods for identifying **modulators** of body **fat metabolism** and the development of treatments for obesity and obesity-related diseases.

L6 ANSWER 4 OF 4 USPATFULL on STN
ACCESSION NUMBER: 2003:245127 USPATFULL
TITLE: G-protein coupled receptor molecules and uses thereof
INVENTOR(S): Elliott, Steven G., Newbury Park, CA, UNITED STATES
Rogers, Norma, Moorpark, CA, UNITED STATES
Busse, Leigh Anne, Camarillo, CA, UNITED STATES
PATENT ASSIGNEE(S): Amgen Inc., A Corporation of the State of Delaware (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003171541	A1	20030911
APPLICATION INFO.:	US 2002-76260	A1	20020214 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-269040P	20010214 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE 3200, CHICAGO, IL, 60606
NUMBER OF CLAIMS: 58
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 4316
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides G-Protein Coupled Receptor (GPCR) polypeptides and nucleic acid molecules encoding the same. The invention also provides selective binding agents, vectors, host cells, and methods

for producing GPCR polypeptides. The invention further provides pharmaceutical compositions and methods for the diagnosis, treatment, amelioration, and/or prevention of diseases, disorders, and conditions associated with GPCR polypeptides.

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	22.73	22.94

STN INTERNATIONAL LOGOFF AT 13:42:16 ON 22 FEB 2006